Public Comment
Proposed Recycled Water Policy Amendment
Deadline: 9/10/18 by 12 noon

RECEIVED
8-28-18
SWRCB Clerk

From: McLellan, Laura@Waterboards
To: commentletters
Subject: FW: COMMENT Concerning PFOS and PFOA
Date: Wednesday, August 29, 2018 1:23:43 PM

Good afternoon,
I received this comment regarding the Proposed Recycled Water Policy Amendment and I believe it should be recorded as a written comment for the current public comment period.
Thank you,

Laura McLellan
Environmental Scientist
Division of Water Quality: Recycled Water, Desalination, and CECs Unit
(916) 319-8288

From: Edo McGowan <edo_mcgowan@hotmail.com>
Sent: Tuesday, August 28, 2018 7:52 AM
To: McLellan, Laura@Waterboards <Laura.McLellan@Waterboards.ca.gov>; JohnAckerman <johnmackerman@gmail.com>; Samantha.Omana@asm.ca.gov; Ruth Ackerman <dr.ruth1232@gmail.com>
Subject: COMMENT Concerning PFOS and PFOA

To: Laura McLellan --- Laura.McLellan@waterboards.ca.gov

Fm: Dr Edo McGowan

Re: NOTICE OF OPPORTUNITY TO COMMENT Concerning PFOS and PFOA Revisions to an Amendment to the Policy for Water Quality Control for Recycled Water

comments on changes to the Revised Proposed Amendment and Revised Draft SR/SED associated with the addition of PFOS and PFOA to the CEC monitoring list, which are identified by underline or strike-through text

COMMENT: Some attention seems warranted with respect to interactions between PFOS and PFOA and their contact with pharmaceuticals as found

discharged into wastewater treatment systems, and subsequent impacts on mitochondria. See below for a start:

Damage to mitochondria is now understood to play a role in the pathogenesis of a wide range of seemingly unrelated disorders such as schizophrenia, bipolar disease, dementia, Alzheimer's disease, epilepsy, migraine headaches, strokes, neuropathic pain, Parkinson's disease, ataxia, transient ischemic attack, cardiomyopathy, coronary artery disease, chronic fatigue syndrome, fibromyalgia, retinitis pigmentosa, diabetes, hepatitis C, and primary biliary cirrhosis.

Mol Nutr Food Res. 2008 Jul;52(7):780-8. doi: 10.1002/mnfr.200700075.

Medication-induced mitochondrial damage and disease.

Neustadt J[1], Pieczenik SR.

Author information

Abstract

Since the first mitochondrial dysfunction was described in the 1960s, the medicine has advanced in its understanding the role mitochondria play in health and disease. Damage to mitochondria is now understood to play a role in the pathogenesis of a wide range of seemingly unrelated disorders such as schizophrenia, bipolar disease, dementia, Alzheimer's disease, epilepsy, migraine headaches, strokes, neuropathic pain, Parkinson's disease, ataxia, transient ischemic attack, cardiomyopathy, coronary artery disease, chronic fatigue syndrome, fibromyalgia, retinitis pigmentosa, diabetes, hepatitis C, and primary biliary cirrhosis. Medications have now emerged as a major cause of mitochondrial damage, which may explain many adverse effects. All classes of psychotropic drugs have been documented to damage mitochondria, as have stain medications, analgesics such as acetaminophen, and many others. While targeted nutrient therapies using antioxidants or their precursors (e. g., N-acetylcysteine) hold promise for improving mitochondrial function, there are large gaps in our knowledge. The most rational approach is to understand the mechanisms underlying mitochondrial damage for specific medications and attempt to counteract their deleterious effects with nutritional therapies. This article reviews our basic understanding of how mitochondria function and how medications damage mitochondria to create their occasionally fatal adverse effects.

Toxicol Sci. 2004 Nov;82(1):333-40. Epub 2004 Aug 13.

Mitochondrial permeability transition as the critical target of N-acetyl perfluorooctane sulfonamide toxicity in vitro.

O'Brien TM[1], Wallace KB.

Author information

Abstract

Perfluorooctanyl compounds with active functional groups have been shown to disrupt mitochondrial bioenergetics by three distinct mechanisms: protonophoric uncoupling of mitochondrial respiration, induction of the mitochondrial permeability transition (MPT), or a nonselective increase in membrane permeability. The purpose of this investigation was to identify the initial target and specific sequence of events associated with the N-acetyl substituted perfluorooctanesulfonamides induced MPT. N-acetyl-perfluorooctanesulfonamide (FOSAA), N-ethyl-N-acetyl-perfluorooctanesulfonamide (N-Et FOSAA), perfluorooctanoic acid (PFOA), perfluorooctanesulfonate (PFOS), and N-ethyl-N-(2-ethoxy)-perfluorooctanesulfonamide (N-Et FOSE) were added individually to liver mitochondria freshly isolated from Sprague-Dawley rats. Mitochondrial swelling and cytochrome c release were recorded spectrophotometrically, oxygen uptake was monitored with a Clark-type oxygen electrode, and reactive oxygen species (ROS) were monitored by dichlorodihydrofluorescein diacetate (H(2)DCFDA) fluorescence. FOSAA (45 microM) and N-Et FOSAA (7.5 microM) induced calcium-dependent mitochondrial swelling, the release of cytochrome c, inhibition of uncoupled mitochondrial respiration, and ROS generation, all of which were inhibited by cyclosporin-A (CsA). PFOA (200 microM) displayed slight CsA sensitive activity, but neither PFOS (10 microM) nor N-Et FOSE (70 microM) induced the MPT. Results of this investigation demonstrate two important findings: (1) MPT induction is specific to the N-acetyl substituted perfluorooctanesulfonamides and, (2) the sequence of events is initiated by induction of the MPT, which causes the release of cytochrome c as well as other cofactors leading to inhibition of respiration and ROS generation. The toxicity of N-acetyl perfluorooctanyl compounds may therefore reflect the mitochondrial dysfunction, which is compounded by the ensuing oxidative injury.

PMID:15310855DOI:10.1093/toxsci/kfh244

J Hazard Mater. 2012 Jan 30;201-202:209-18. doi: 10.1016/j.jhazmat.2011.11.061. Epub 2011 Nov 29.

Toxicological interactions of

perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) with selected pollutants.

Rodea-Palomares I[1], Leganés F, Rosal R, Fernández-Piñas F.

Author information

Abstract

The combined toxicity of the perfluorinated surfactants perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA) and several pollutants (Hg(2+), Cd(2+), 2,4-D, propylparaben, mitomycin C and furazolidone) has been examined with a bioluminescent cyanobacterial toxicity test. Hg(2+), Cd(2+), mitomycin C and furazolidone could be included in the "Acute aquatic hazard" category established in the Regulation (EC) No 1272/2008 being "very toxic to aquatic life". Toxicological interactions of PFOA, PFOS with these pollutants in binary, ternary and multicomponent mixtures were studied using the combination-index method. PFOA and PFOS showed an antagonistic interaction at the whole range of effect levels, this may explain in part the finding that PFOA and PFOS interacted in an inverse way with the organic pollutants; the relative hydrophobicity of the tested compounds would also explain this interaction pattern. The interaction of both PFOS and PFOA with heavy metals was mostly antagonistic, decreasing metal toxicity. With increasing complexity of the mixtures, the CI method predicted synergism at low to very low levels of effect; pollutant combinations at their mixture NOECs were tested and confirmed the predicted synergism.

Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA ...

https://www.ncbi.nlm.nih.gov/pubmed/19359027

by J Yu - 2009 - Cited by 189 - Related articles

Mar 18, 2009 - *Perfluorooctane sulfonate* (*PFOS*) and *perfluorooctanoic acid* (*PFOA*) in *sewage* treatment plants. Yu J(1), Hu J, Tanaka S, Fujii S.

Fluorinated alkyl surfactants

MM Schultz, DF Barofsky, JA Field - Environmental Engineering …, 2003 - liebertpub.com

… 9. Urban **effluent discharges** as causes of public and environmental health concerns … Fluorotelomer
sulfonate aerobic biotransformation in activated sludge of **waste water** treatment plants … and
behavior of perfluorinated compounds (PFCs) in several **wastewater** treatment plants …

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Comparison of *in vitro* cytotoxicity, estrogenicity and anti-estrogenicity of

triclosan, perfluorooctane sulfonate and perfluorooctanoic acid

ND Henry, PA Fair - Journal of Applied Toxicology, 2013 - Wiley Online Library

… PPCPs, PFCs may enter the environment through **wastewater** treatment **discharge**; however,
despite … 1, Concentration in Charleston, SC area **wastewater** treatment plant **effluent** (Fair et … mean
concentrations of triclosan entering the environment from **wastewater** treatment plants …

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